NEUROLOGIA I NEUROCHIRURGIA POLSKA 50 (2016) 280–283

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Case report

# Acute infarction of corpus callosum due to transient obstructive hydrocephalus

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ARTICLE INFO

*Article history:*
Received 23 January 2016
Accepted 14 March 2016
Available online 23 March 2016

*Keywords:*
Corpus callosum
Hydrocephalus
Ischemia
Diffuse axonal injury

ABSTRACT

Acute ischemia of the corpus callosum (CC) is not a well-known feature in patients with acute hydrocephalus. Herein, we describe a case with acute CC infarction due to another rare entity; transient obstructive hydrocephalus. A 66-year-old male was admitted with sudden onset right-sided hemiparesia. CT demonstrated a hematoma on the left basal ganglia with extension to all ventricles. The following day, the patient's neurological status progressed to coma and developed bilateral pyramidal signs. MRI demonstrated obstructive hydrocephalus and acute diffuse infarction accompanied by elevation of the CC. On the same day there was improvement in his neurological status with significant decrease in ventricular size and complete resolution of the clot in the third ventricle. The mechanism of signal abnormalities is probably related with the neural compression of the CC against the falx. Presumably, the clot causing obstruction in the third ventricle dissolved or decayed by the help of fibrinolytic activity of CSF, which was raised after IVH and caused spontaneous improvement of hydrocephalus. Bilateral neurological symptoms suggest diffuse axonal damage and normalization of the intracranial pressure should be performed on the early onset of clinical detorioration in order to prevent axonal injury.



## 1. Introduction

Arterial supply of corpus callosum (CC) is derived from both anterior and posterior circulation.

Therefore, infarcts of CC are not common and mainly caused by infarctions of anterior cerebral artery and posterior cerebral artery territories. Because of the rich blood supply, solitary infarct of the CC is rare and generally associated with infarcts of the neighboring structures [1]. Morphological changes such as elevation, thinning and impingement of CC have previously been described in acute hydrocephalus whereas acute ischemia is not a well-known feature [2–4]. Herein, we describe a case with acute CC infarction due to another rare entity; transient posthemorrhagic obstructive hydrocephalus.

## 2. Case report

A 66-year-old male was admitted to our hospital with the chief complain of sudden onset of right sided hemiparesia. He had hypertension in his past medical history. On neurological examination, he was alert with full cooperation and orientation,

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http://dx.doi.org/10.1016/j.pjnns.2016.03.005

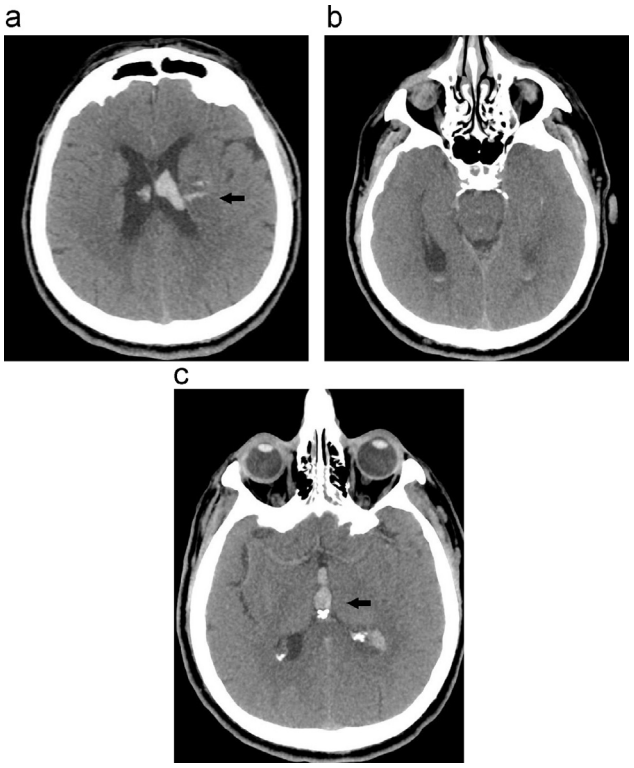


**Fig. 1 – The brain CT performed in emergency service shows a left sided basal ganglia hematoma (a) with extension to all ventricles (b) and the clot in the third ventricle (c).**

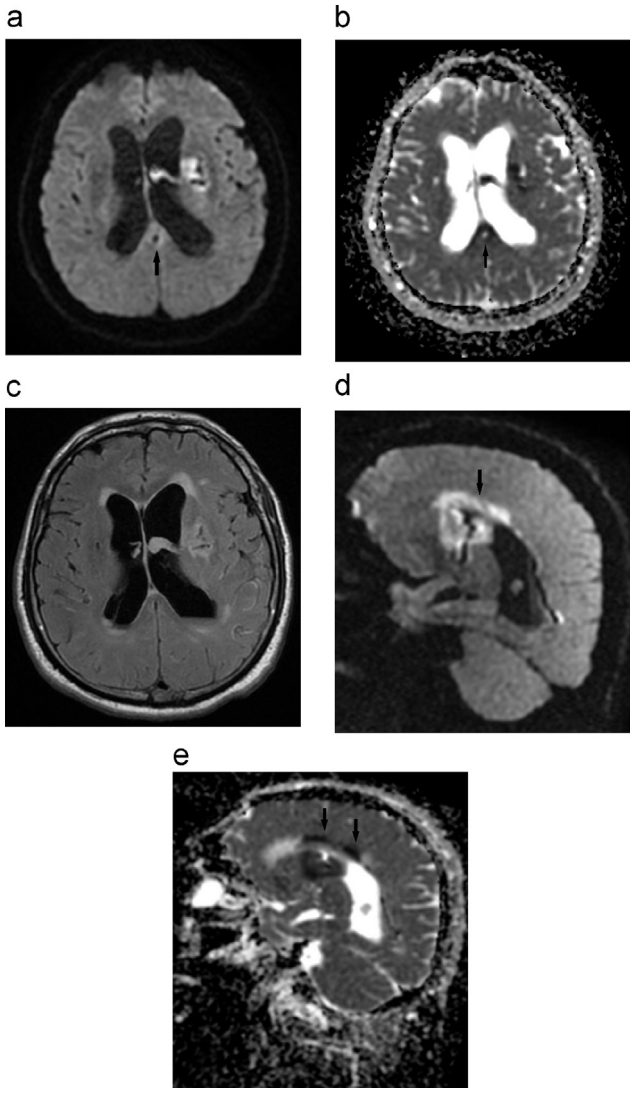


**Fig. 2 – Axial diffusion (a), apparent diffusion coefficient (ADC) (b), sagittal diffusion (d) sagittal ADC (e) sequences of the brain MRI demonstrate hemorrhage in left basal ganglia and the infarction of the CC. The fluid-attenuated inversion recovery (FLAIR) sequence does not show any signal changes (c) in CC indicating hyperacute phase of ischemia. Elevation of the CC was seen in the sagittal sequences.**

he had right sided central facial palsy, hemiparesia, hemihypoesthesia and the Babinski sign. The computerized tomography (CT) of the brain showed a left sided basal ganglia hematoma with extension to all ventricles, the volume of intraventricular hemorrhage was 9 ml (Fig. 1). The patient was hospitalized and followed in the intensive care unit. Twenty-two hours after presentation, he became stuporous and CT showed ventricular enlargement. Four hours later he underwent brain magnetic resonance imaging (MRI) to exclude other reasons of clinical impairment. MRI demonstrated acute obstructive hydrocephalus, elevation and hyperacute infarction of the corpus callosum (Fig. 2). He was intubated; hyperventilation and mannitol therapy was started. The neurological status progressed to coma within hours and he developed extensor response to noxious stimuli and bilateral Babinski sign. Although there was no cause explaining neurological impairment other than hydrocephalus, the neurosurgery department recommended following the patient with medical therapy and did not perform any intervention. Sixteen hours later, neurological deterioration suddenly ceased and later on he was able to localize painful stimuli. The CT obtained the next day, 48 h after initial presentation showed significant decrease in the ventricular size and complete resolution of the clot in the third ventricle (Fig. 3). The CC infarction was more prominent on the MRI which was obtained 12 days after the incident (Fig. 4). Digital subtraction angiography did not reveal any vascular pathology. Consequently, the etiology of the bleeding was considered as hypertensive hemorrhage. He was discharged two months later and was able to cooperate to all simple and some complex commands and his muscle strength had recovered to grade 4/5 in all four limbs.

## 3. Discussion

We described a recent case of acute infarction of the CC due to acute obstructive hydrocephalus. The subcallosal and medial callosal arteries supply blood to the anterior part of the CC, the pericallosal artery supplies the body, while the posterior pericallosal artery is responsible for the blood supply of the splenium. Because of its rich blood supply, the CC is known as a resistant area for ischemia. Hence isolated callosal infarcts

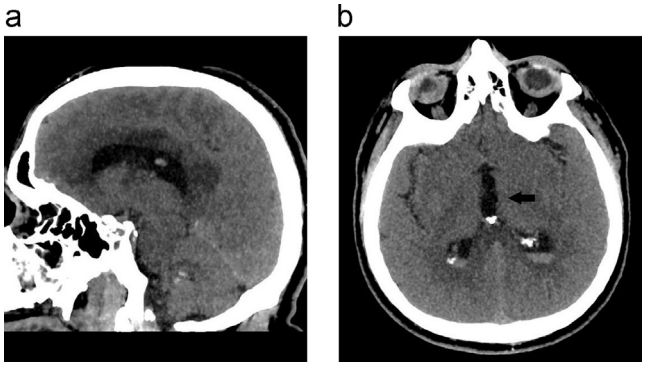


**Fig. 3 – The CT performed when the neurological improvement started (48 h after initial presentation) shows significant decrease in the ventricular size (a) and complete resolution of the clot in the third ventricle (b).**

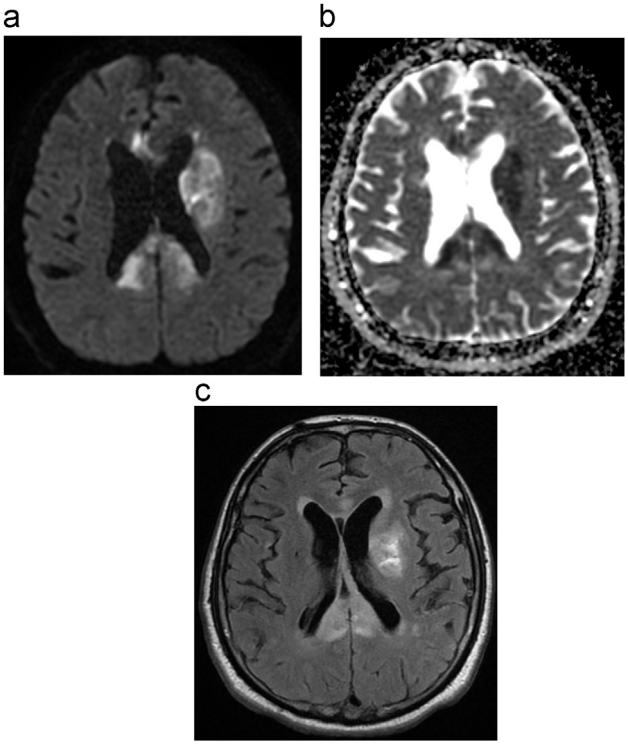


**Fig. 4 – Axial diffusion (a), apparent diffusion coefficient (ADC) (b) and FLAIR (c) sequences demonstrate the subacute infarction of CC in the follow-up MRI.**

are very uncommon. If present, they affect the splenium more often than the body and genu. They usually accompany larger territorial infarcts. Diffuse ischemia of CC has been described in patients after status epilepticus and cardiopulmonary arrest [5].

Morphological changes in CC may present in obstructive hydrocephalus with or without shunting. Elevation, thinning, impingement of CC may occur in active hydrocephalus whereas scalloping deformity and generalized thickening has been observed after shunting [2,4,6]. Signal changes are described both before and after shunting [4,7]. It has been mentioned that the changes detected after shunting are in fact associated with hydrocephalus but become visible only after ventricular decompression [8]. In our patient, the mechanism of CC infarction in acute hydrocephalus is probably related with the neural compression of CC. The upward displacement of CC secondary to third and lateral ventriculomegaly causes stretching of the corpus callosum against the rigid falx. Impairment of the venous drainage or arterial supply could lead to ischemia. Numaguchi et al. observed decreased signal intensity on T1-weighted images (did not report T2-weighted images) in some of their patients with hydrocephalus. Because the low intensity disappeared or became less distinct on follow-up MR images, they pointed out that callosal signal changes could be temporary. They noted that permanent damage, such as atrophy of the corpus callosum, occurs due to long-term hydrocephalus [6]. In our patient, CC infarction was detected on the MRI scan performed soon after the significant onset of clinical deterioration. Since the CC infarction occurred in the early phase of hydrocephalus, we believe that the rapid increase in the intracranial pressure could also play an important role in the mechanism of CC lesions. It can debated that if the interventions reducing the hydrocephalus were applied at an early period, the signal changes would be reversible.

The other interesting entity of our case is transient obstructive hydrocephalus. Obstructive hydrocephalus is common after intraventricular hemorrhage (IVH) due to obstruction of normal CSF flow. Spontaneous resolution of acute hydrocephalus without drainage of cerebrospinal fluid (CSF) is rare, and there are only a few case reports in the literature up to date [9–12]. Our patient had obstructive hydrocephalus secondary to the clot in the aqueduct. Presumably, the clot caused obstruction of third ventricle in our case dissolved or decayed by the help of the fibrinolytic activity of CSF, which was raised after IVH.

Another point is that the CC lesions probably provided an additional contribution to the impairment of motor and cognitive functions of the patient. The addition of ipsilateral pyramidal findings in the period of hydrocephalus suggests the presence of diffuse axonal injury. Since the bilateral neurological symptoms are disproportionate to imaging findings, the degree of axonal injury is considered to be microscopic. It was stated that the best approach in such patients would be to closely monitor the clinical status, and ventricular drainage or endoscopic removal of the clot should be performed in case of any deterioration in the level of consciousness [11]. The impairment of the neurological status of our case due to axonal injury could have been prevented by the normalization of the intracranial pressure.

## 4. Conclusion

In summary, as well as morphological changes, the infarct of CC may be seen as a feature of acute obstructive hydrocephalus. Posthemorrhagic hydrocephalus could be transient due to the movement or dissolving of the clot causing occlusion. Normalization of the intracranial pressure should be performed in the early stage in the presence of clinical deterioration in order to prevent axonal injury.

## Conflict of interest

None declared.

## Acknowledgement and financial support

None declared.

## Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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